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FILE LAST UPDATED: 27 Mar 2008  (20080327/ED)

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  This file contains CAS Registry Numbers for easy and accurate
  substance identification.


=> s adiponectin or apM1
         3870 ADIPONECTIN
          159 APM1
L1       3952 ADIPONECTIN OR APM1

=> s l1 and (monoclonal or polyclonal)
       149693 MONOCLONAL
        37600 POLYCLONAL
L2         33 L1 AND (MONOCLONAL OR POLYCLONAL)

=> s l2 and latex particles
        72295 LATEX
       825586 PARTICLES
         6918 LATEX PARTICLES
                (LATEX(W)PARTICLES)
L3          1 L2 AND LATEX PARTICLES

=> d ti ab

L3   ANSWER 1 OF 1  CA  COPYRIGHT 2008 ACS on STN
TI   Latex reagent for   ***adiponectin***   analysis, and   ***adiponectin***
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analysis method

AB A latex reagent for ***adiponectin*** anal. is provided, which comprises a suspension of ***latex*** ***particles*** carrying a substance (e.g., anti- ***adiponectin*** ***polyclonal*** antibody) capable of specifically binding with ***adiponectin*** . Also provided is a method for ***adiponectin*** anal., which comprises: (1) a step for obtaining a biol. liq. potentially contg. ***adiponectin*** ; and (2) a step for contacting the biol. liq. obtained in the step (1) as it is with a suspension of ***latex*** ***particles*** carrying a substance capable of specifically binding with ***adiponectin*** , and optically analyzing the resultant mixt. to det. the degree of agglutination of the ***latex*** ***particles*** . According to this latex reagent for ***adiponectin*** anal. and this ***adiponectin*** anal. method, it is not required to dil. or pretreat a biol. liq. as a test sample beforehand, and the anal. is rapidly and conveniently performed without limiting a measurement facility.

=> d all

L3 ANSWER 1 OF 1 CA COPYRIGHT 2008 ACS on STN
AN 141:310247 CA <<LOGINID::20080330>>
ED Entered STN: 28 Oct 2004
TI Latex reagent for ***adiponectin*** analysis, and ***adiponectin*** analysis method
IN Tachikawa, Tetsuya; Akamatsu, Suguru; Sawai, Tokio; Nishimura, Fumiko
PA Mitsubishi Kagaku Iatron, Inc., Japan; Otsuka Pharmaceutical Co., Ltd.
SO PCT Int. Appl., 26 pp.
CODEN: PIXXD2
DT Patent
LA Japanese
IC ICM G01N033-53
ICS G01N033-543
CC 9-10 (Biochemical Methods)
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004086040	A1	20041007	WO 2004-JP4083	20040324
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2004223553	A1	20041007	AU 2004-223553	20040324
	CA 2520438	A1	20041007	CA 2004-2520438	20040324
	EP 1607742	A1	20051221	EP 2004-723044	20040324
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK				
	US 20070037207	A1	20070215	US 2005-550324	20050923
PRAI	JP 2003-80763	A	20030324		

WO 2004-JP4083 W 20040324

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 2004086040	ICM	G01N033-53
	ICS	G01N033-543
	IPCI	G01N0033-53 [ICM,7]; G01N0033-543 [ICS,7]
	IPCR	G01N0033-53 [I,C*]; G01N0033-53 [I,A]; G01N0033-543 [I,C*]; G01N0033-543 [I,A]; G01N0033-68 [I,C*]; G01N0033-68 [I,A]
	ECLA	G01N033/543D; G01N033/68V
AU 2004223553	IPCI	G01N0033-53 [ICM,7]; G01N0033-543 [ICS,7]
	IPCR	G01N0033-53 [I,C*]; G01N0033-53 [I,A]; G01N0033-543 [I,C*]; G01N0033-543 [I,A]; G01N0033-68 [I,C*]; G01N0033-68 [I,A]
CA 2520438	IPCI	G01N0033-53 [ICM,7]; G01N0033-543 [ICS,7]
	IPCR	G01N0033-53 [I,C*]; G01N0033-53 [I,A]; G01N0033-543 [I,C*]; G01N0033-543 [I,A]; G01N0033-68 [I,C*]; G01N0033-68 [I,A]
	ECLA	G01N033/543D; G01N033/68V
EP 1607742	IPCI	G01N0033-53 [ICM,7]; G01N0033-543 [ICS,7]
	IPCR	G01N0033-53 [I,C*]; G01N0033-53 [I,A]; G01N0033-543 [I,C*]; G01N0033-543 [I,A]; G01N0033-68 [I,C*]; G01N0033-68 [I,A]
	ECLA	G01N033/543D; G01N033/68V
US 20070037207	IPCI	G01N0033-53 [I,A]; G01N0033-551 [I,A]
	NCL	435/007.100; 436/524.000

AB A latex reagent for ***adiponectin*** anal. is provided, which comprises a suspension of ***latex*** ***particles*** carrying a substance (e.g., anti- ***adiponectin*** ***polyclonal*** antibody) capable of specifically binding with ***adiponectin*** . Also provided is a method for ***adiponectin*** anal., which comprises: (1) a step for obtaining a biol. liq. potentially contg. ***adiponectin*** ; and (2) a step for contacting the biol. liq. obtained in the step (1) as it is with a suspension of ***latex*** ***particles*** carrying a substance capable of specifically binding with ***adiponectin*** , and optically analyzing the resultant mixt. to det. the degree of agglutination of the ***latex*** ***particles*** . According to this latex reagent for ***adiponectin*** anal. and this ***adiponectin*** anal. method, it is not required to dil. or pretreat a biol. liq. as a test sample beforehand, and the anal. is rapidly and conveniently performed without limiting a measurement facility.

ST ***adiponectin*** analysis latex agglutination reagent antibody

IT Antibodies and Immunoglobulins
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(***adiponectin*** anal. method using latex agglutination immunoassay reagent)

IT Cytokines
RL: ANT (Analyte); ANST (Analytical study)
(***adiponectin*** ; ***adiponectin*** anal. method using latex agglutination immunoassay reagent)

IT Agglutination test
(latex; ***adiponectin*** anal. method using latex agglutination immunoassay reagent)

IT ***Latex***
(***particles*** ; ***adiponectin*** anal. method using latex

agglutination immunoassay reagent)
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) A&t Corp; JP 20014624 A 2001
(2) Otsuka Pharmaceutical Co Ltd; EP 1033134 A 1999 CA
(3) Otsuka Pharmaceutical Co Ltd; US 6461821 B 1999 CA
(4) Otsuka Pharmaceutical Co Ltd; WO 9921577 A 1999 CA

=> d l2 ti ab 1-33

L2 ANSWER 1 OF 33 CA COPYRIGHT 2008 ACS on STN
TI CNGH0010-specific polynucleotides, polypeptides, antibodies, compositions, methods and uses
AB Novel polypeptides (CNGH0010) and antibodies, including specified portions or variants, specific for at least one such CNGH0010 polypeptide, variant, or fragment thereof, as well as nucleic acids encoding such CNGH0010 polypeptides and antibodies, complementary nucleic acids, vectors, host cells, and methods of making and using thereof, are useful for therapeutic and diagnostic formulations, administration and devices. At least six spliced variants of the CNGH0010 polypeptides are identified. The aforesaid polypeptides can be used to generate human, primate, rodent, mammalian, chimeric, humanized, and/or CDR-grafted anti-CNGH0010 antibodies. The similarities of the CNGH0010 mols. to extracellular matrix mols. (collagens) and cytokines (***adiponectin***) suggest that they may participate in the regulation of cell-cell or cell-matrix interactions, or bind to or influence the binding of a cytokine to a receptor. Transcript levels in selected disease tissues also suggest that the CNGH0010 polypeptides and antibodies are useful in modulating or treating at least one CNGH0010-related disease in a cell, tissue, organ, animal, or patient. Such diseases may include, but are not limited to, psoriasis, rheumatoid arthritis, emphysema, asthma, diabetes, autoimmune thyroiditis, inflammatory bowel diseases, including Crohn's disease and ulcerative colitis, different types of dermatitis including allergic dermatitis, contact dermatitis, actinic keratosis, wound healing, scar formation, various renal diseases, various respiratory diseases, various diseases of reproductive organs, such as endometriosis, melanoma, squamous cell carcinoma, ovarian cancer, breast cancer, lung cancer, colon cancer, prostate cancer, renal cell carcinoma, Grave's disease, and other inflammatory and hyperproliferative diseases.

L2 ANSWER 2 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Quickly detecting the content of plasma Acrp30 by competitive ELISA
AB Here, full- length ***adiponectin*** (Acrp30) and its C-terminal globular head domain (gAcrp30) were expressed in Escherichia coli and gAcrp30 was used to immunize a rabbit to obtain ***polyclonal*** antiserum with titer of 10000. Competitive Enzyme - Linked Immunosorbent Assay (ELISA) was used in the expt., the std. antigen was covered in each well, secondly, the antibody of gAcrp30 was mixed with std. antigen (as a stayer) or samples for 30 min, then the mixt. was added to each well, after that the second antibody was added, finally TMD added, after the reaction was stopped by 2 mol/L H2SO4, read at 450 nm immediately. A std. graph was plotted by using the logarithm of std. antigen dild. times as X - axis, and the OD450 as Y - axis. The content of plasma sample could be detected with the graph. A method of quickly detecting the content of plasma Acrp30 is founded.

L2 ANSWER 3 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Detection of soluble ***adiponectin*** receptor peptides and use in diagnostics and therapeutics
AB The present invention relates to sol. C-terminal fragments of the ***adiponectin*** receptor and their use in the diagnosis and management of disorders involving adipocyte imbalance. Accordingly, the present invention provides, among other thing, the fragments, methods of detecting them, methods of using them and antibodies capable of binding to them.

L2 ANSWER 4 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Development of an ELISA kit for mouse and rat visfatin
AB A review discussing (1) recombinant visfatin and anti-visfatin ***monoclonal*** antibody, (2) ELISA kit for detection of visfatin, (3) std. curve, (4) diln. study, (5) reproducibility, (6) sample stability and (7) visfatin and ***adiponectin*** expression in adipocytes.

L2 ANSWER 5 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Characterization of recombinant human globular domain of ***adiponectin***
AB CDNA encoding human globular domain of ***adiponectin*** (gAPN) was amplified by PCR, and cloned in the expression vector pET28a(+). The recombinant expression plasmid pET28a(+)-gAPN was transformed into E. coli BL21 (DE3). When A600 (nm) reached 0.6, this transformant was induced for expression of recombinant protein by isopropyl-.beta.-D-thiogalactopyranoside (IPTG). Recombinant protein was obtained after denaturation, purifn. by Ni+-affinity chromatog., and renaturation. The bioactivity of the recombinant protein was evaluated by Streptozocin (STZ)-induced hyperglycemia model. After induction by IPTG for 3 h, the recombinant protein was over 30% of total bacterial protein. The recombinant protein was expressed in form of inclusion bodies. After purifn., SDS-PAGE anal. indicated that the mol. wt. of the recombinant protein was about 17000 and the purity was up to 90%. Western blot indicated that the recombinant protein could react with anti-APN ***polyclonal*** antibody. The recombinant protein could significantly reduce the level of blood glucose in STZ-induced hyperglycemia model. GAPN is successfully expressed in E. coli BL21 (DE3) and the bioactive protein is also obtained.

L2 ANSWER 6 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Observation and analysis of Lp(a), ***adiponectin*** and TNF-.alpha. in patients with mammary adenocarcinoma
AB The objective is to study changes and clin. significances of plasma Lp(a), serum ***adiponectin*** and TNF-.alpha. in patients with mammary adenocarcinoma. ***Monoclonal*** antibody turbidimetry and ELISA methods were used to detect the plasma Lp(a), serum ***adiponectin*** and TNF- .alpha. in 35 patients with mammary adenocarcinoma before and after operation and 38 healthy women. Plasma Lp(a) and serum TNF-.alpha. in patients with mammary adenocarcinoma before operation were higher but serum ***adiponectin*** was lower than that in healthy women (P <0.01). Plasma Lp(a) and serum TNF-.alpha. were got closure to the control group after operation and had significant difference compared to before operation. Serum ***adiponectin*** was increased in patients but had no significance with that before operation. Serum ***adiponectin*** had significant relation (r = 0.578, P <0.01) before operation but disappeared after operation. Plasma Lp(a) and serum ***adiponectin*** , TN-.alpha. may play important roles in patients with

mammary adenocarcinoma.

L2 ANSWER 7 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Cloning and soluble expression of ***adiponectin*** and gadiponectin in E. coli
AB Objectives: To clone and express ***adiponectin*** and gadiponectin with biol. activities in E. coli expression system and prep. anti-***adiponectin*** and anti-gadiponectin ***polyclonal*** antibodies.
Methods: The ***adiponectin*** and gadiponectin-coding DNAs were amplified from pQE30- ***adiponectin*** plasmid by PCR. Then the genes were subcloned into pGEX-4T-2 vector to construct recombinant expression plasmids and transformed into competent host E. coli BL21 to induce and express fusion proteins. The sol. proteins were purified by GSTrap affinity chromatog. The specificity of the ***polyclonal*** antibody prepd. was examd. by Western blot. Results: 700 bp ***adiponectin*** and 400 bp gadiponectin DNA fragments were amplified from pQE30-***adiponectin*** plasmids by PCR. The mol. wt. of GST-***adiponectin*** and GST-gadiponectin recombinant fusion proteins were about 51 kD and 42 kD, resp. After purifn., the purity was over 90%. The specificity of the ***polyclonal*** antibodies prepd. with serum ***adiponectin*** were confirmed by Western blot. Conclusion: The expressed proteins and antibodies prepd. may be used for detection and functional anal. of ***adiponectin*** .

L2 ANSWER 8 OF 33 CA COPYRIGHT 2008 ACS on STN
TI A novel ELISA system for selective measurement of human ***adiponectin*** multimers by using proteases
AB Background: ***Adiponectin*** , an antiatherogenic adipocyte-derived protein exists in human blood as multiple isoforms-trimeric low mol. wt. (LMW), albumin-binding LMW (Alb-LMW), hexameric middle mol. wt. (MMW), and high mol. wt. (HMW) forms. The authors developed a novel ELISA system to detect total human ***adiponectin*** and the selective level of each ***adiponectin*** multimer for investigating the distribution of these levels in human blood. Methods: Two ***monoclonal*** antibodies that were raised against human ***adiponectin*** were used to construct a sandwich ELISA to measure ***adiponectin*** levels. ***Adiponectin*** multimers were selectively measured after sample pretreatment with two proteases that specifically digested the trimeric forms or both the hexameric and trimeric forms. Results: The ELISA had a dynamic range of 0.075-4.8 ng/mL. Intraassay variations (CV) were 5.3% (total ***adiponectin***), 4.1% (MMW + HMW), and 3.3% (HMW). Comparison of the results of ELISA and quant. western blot anal. of multimeric ***adiponectin*** in serum samples revealed good correlation (LMW + Alb-LMW, r = 0.873; MMW, r = 0.907; HMW, r = 0.950). Each of the three forms of ***adiponectin*** multimer levels closely correlated with total ***adiponectin*** levels in healthy subjects. Conclusions: This ELISA system can be used to further investigate the physiol. roles of human ***adiponectin*** multimers.

L2 ANSWER 9 OF 33 CA COPYRIGHT 2008 ACS on STN
TI A novel enzyme-linked immunosorbent assay specific for high-molecular-weight ***adiponectin***
AB Human plasma contains at least three forms of ***adiponectin*** : a trimer, a hexamer, and a high-mol.-wt. (HMW) multimer. We purified HMW ***adiponectin*** from human plasma using its affinity to gelatin and obtained ***monoclonal*** antibodies against it. On Western blot

anal., the reactivity of these ***monoclonal*** antibodies was shown to be restricted to a non-heat-denatured form of ***adiponectin*** mols. On heating, the collagen-like domain of ***adiponectin*** mols. became denatured, and thus the trimer form could not be maintained. From these, ***monoclonal*** antibodies against HMW ***adiponectin*** were suggested to react with the intact trimer of ***adiponectin*** . With these ***monoclonal*** antibodies, we developed a sandwich ELISA system for quantifying ***adiponectin*** in human serum. Its specificity was verified by anal. of serum fractions sepd. by gel-filtration chromatog., and our ELISA system was found to be HMW ***adiponectin*** -specific. With this novel ELISA, the HMW ***adiponectin*** concns. were 8.4 .+-. 5.5 .mu.g/mL (mean .+-. SD) in healthy women and 6.2 .+-. 3.6 .mu.g/mL in healthy men. Also, serum with a lower HMW ***adiponectin*** concn. was shown to have a lower HMW ratio (i.e., HMW ***adiponectin*** /total ***adiponectin***).

L2 ANSWER 10 OF 33 CA COPYRIGHT 2008 ACS on STN

TI Determination of ***adiponectin*** in serum using a latex particle-enhanced turbidimetric immunoassay with an automated analyzer

AB ***Adiponectin*** is an adipose-derived hormone that plays a role in regulating metabolic processes such as fat partitioning and lipid and glucose metab. Quantification of ***adiponectin*** is useful for obtaining information on metabolic syndrome, but there is no rapid method to measure ***adiponectin*** for clin. use. The authors developed a rapid and sensitive latex particle-enhanced turbidimetric immunoassay (LTIA) using a latex bead-immobilized anti- ***adiponectin*** ***polyclonal*** antibody. The assay was performed on a Hitachi H7170 analyzer and evaluated for validity as a method to quantitate ***adiponectin*** , in parallel with the ELISA. Diln. tests using LTIA showed linearity from 0.25 to 30 .mu.g/mL. Within-run CV and total CV were obtained in the range 0.8-1.9% and 1.1-2.0%, resp. No interference was obsd. in the testing of specimens contg. potentially interfering substances such as bilirubin, ditaurobilirubin, Hb triglyceride, rheumatoid factor, type IV collagen, fibronectin, and complement factor (C1q). A strong correlation between LTIA and ELISA was confirmed (n = 30, r = 0.990, y = 0.95x + 0.39). The LTIA assay is applicable to quantitating the serum concn. of ***adiponectin*** . This assay is more convenient and faster than ELISA and suitable for clin. routine anal.

L2 ANSWER 11 OF 33 CA COPYRIGHT 2008 ACS on STN

TI Regulated expression of putative membrane progestin receptor homologues in human endometrium and gestational tissues

AB Rapid non-genomic actions of progesterone are implicated in many aspects of female reprodn. Recently, three human homologs of the fish membrane progestin receptor (mPR) have been identified. We combined bioinformatic anal. with expression profiling to define further the role of these mPRs in human reproductive tissues. Sequence anal. confirmed that the mPRs belong to a larger, highly conserved family of proteins, termed 'progestin and ***adiponectin*** receptors' (PAQRs). A comparison of the expression of mPR transcripts with that of two related PAQR family members, PAQRIII and PAQRIX, in cycling endometrium and pregnancy tissues revealed markedly divergent expression levels and profiles. For instance, endometrial expression of mPR.alpha. and .gamma. and PAQRIX was cycle-dependent whereas the onset of parturition was assocd. with a marked redn. in myometrial mPR.alpha. and .beta. transcripts. Interestingly, mPR.alpha. and PAQRIX were most highly expressed in the placenta, and the tissue expression levels of both genes correlated inversely with that of

the nuclear PR. Phylogenetic anal. demonstrated that PAQRIX belongs to the mPR subgroup of proteins. We also validated a ***polyclonal*** antibody raised against the carboxy-terminus of human mPR.alpha.. Immunohistochem. anal. demonstrated more intense immunoreactivity in placental syncytiotrophoblasts than in endometrial glands or stroma. The data suggest important functional roles for mPR.alpha., and possibly PAQRIX, in specific reproductive tissues, particularly those that express low levels of nuclear PR.

L2 ANSWER 12 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Methods of diagnosing cardiovascular disease by detecting a resistin gene product
AB The invention relates to the diagnosis of vascular disease or aiding in the diagnosis of an existing vascular condition or in detg. the risk that an individual without a vascular condition has of developing such a condition in the future, and methods of assessing if an individual is at risk of developing a vascular event. The invention relates to predicting which individuals are at risk of developing atherosclerotic vascular disease, and once having disease, which individuals are at risk of experiencing plaque rupture which, depending on the site of the plaque, could produce myocardial infarction, stroke, crit. limb ischemia, or other vascular event. The invention further relates to methods of diagnosing and aiding in the diagnosis of vascular conditions such as atherosclerosis, premature coronary artery disease and coronary artery disease, by detecting a resistin gene product in an individual. The invention further relates to methods of predicting, and aiding in predicting, the likelihood that an individual will experience a vascular event, such as but not limited to, a myocardial infarction, acute coronary syndrome, stroke, transient ischemic attack (TIA), or crit. limb ischemia. The present invention makes it possible to identify individuals at increased risk of developing vascular events, such as but not limited to, strokes and myocardial infarction, and provide treatment to prevent such events or reduce their severity.

L2 ANSWER 13 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Antibody specific to mammalian endogenous ligand without neutralizing activity for stabilizing ligand and enhancing receptor activity to treat diseases
AB An ameliorating agent for the stability of mammalian endogenous ligand in the blood, comprising an antibody having affinity with mammalian endogenous ligand and substantially not neutralizing the same; and prepns. thereof for the prevention and treatment of diseases in accomplishment of which it is effective to increase the concn. of endogenous ligand in the blood and/or prolong the half life period thereof in the blood. When the prepns. alone without being combined with a compd. identical with or substantially identical with the endogenous ligand are administered to a mammal, the stability of endogenous ligand in the blood would be enhanced to thereby reinforce the receptor activity regulating action thereof. The endogenous ligand belonging to the secretin/glucagon superfamily is selected from GLP-1, calcitonin, PACAP, VIP, LHRH, metastin, GPR7/GPR8 ligand, MSH, ghrelin, apelin, EPO, TPO, insulin, interferon, growth hormone, GM-CSF, leptin, ***adiponectin*** , ANP, BNP, CNP, betacellulin, betacellulin-.gamma.4, adrenomedullin.

L2 ANSWER 14 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Rationally designed or domain-exchanged antibodies comprising biologically active peptide for diagnostic or therapeutic purpose

AB Domain-exchanged antibodies having CDR regions replaced or fused with biol. active peptides are described. Flanking sequences may optionally be attached at one or both the carboxy-terminal and amino-terminal ends of the peptide in covalent assocn. with adjacent framework regions. Compns. contg. such modified domain-exchanged antibodies are useful in therapeutic and diagnostic modalities.

L2 ANSWER 15 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Myostatin inhibits myogenesis and promotes adipogenesis in C3H 10T(1/2) mesenchymal multipotent cells
AB Inactivating mutations of the mammalian myostatin gene are assocd. with increased muscle mass and decreased fat mass; conversely, myostatin transgenic mice that overexpress myostatin in the skeletal muscle have decreased muscle mass and increased fat mass. We investigated the effects of recombinant myostatin protein and antimyostatin antibody on myogenic and adipogenic differentiation of mesenchymal multipotent cells. Accordingly, 10T(1/2) cells were incubated with 5'-azacytidine for 3 d to induce differentiation and then treated with a recombinant protein for myostatin (Mst) carboxy terminal 113 amino acids or a ***polyclonal*** anti-Mst antibody for 3, 7, and 14 d. Cells were also cotransfected with a Mst cDNA plasmid expressing the full-length 375-amino acid protein (pcDNA-Mst375) and the silencer RNAs for either Mst (pSil-Mst) or a random sequence (pSil-RS) for 3 or 7 d, and Mst expression was detd. Adipogenesis was evaluated by quant. image anal. of fat cells before and after oil-red-O staining, immunocytochem. of ***adiponectin*** , and Western blot for CCAAT/enhancer binding protein-.alpha.. Myogenesis was estd. by quant. image anal.-immunocytochem. for MyoD (Myo differentiation protein), myogenin, and myosin heavy chain type II, or by Western blot for myogenin. 5'-Azacytidine-mediated differentiation induced endogenous full-length Mst expression. Recombinant Mst carboxy terminal 113 amino acids inhibited both early and late markers of myogenesis and stimulated both early and late markers of adipogenesis, whereas the antibody against Mst exerted the reverse effects. Myogenin levels at 7 d after transfection of pcDNA-Mst375 were reduced as expected and elevated by pSil-Mst, which blocked efficiently Mst375 expression. In conclusion, myostatin promotes the differentiation of multipotent mesenchymal cells into the adipogenic lineage and inhibits myogenesis.

L2 ANSWER 16 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Use of poly(ADP-ribose) polymerase inhibitors for prevention and treatment of diabetic and insulin resistance complications
AB The present invention provides methods of inhibiting the development or progression of atherosclerotic, microvascular, or neurol. disease due to diabetes or insulin resistance in a mammal, or conditions resulting therefrom. The methods involve specifically inhibiting poly(ADP-ribose) polymerase (PARP) activity or accumulation in the mammal. Also provided are antibodies that specifically react with N.alpha.-acetyl-N.delta.(5-hydro-5-methyl)-4-imidazolone. Addnl., the invention provides methods of monitoring the effectiveness of an anti-diabetic or anti-insulin resistance treatment or an anti-diabetic or anti-insulin resistance complication treatment in a mammal. The methods involve measuring ADP-ribosylated protein levels, or measuring methylglyoxyl AGE levels in the mammal using an antibodies that specifically react with N.alpha.-acetyl-N.delta. (5-hydro-5-methyl)-4-imidazolone, or measuring GlcNAc-modified protein levels in the mammal. The present invention is based in part on the discovery that hyperglycemia-induced mitochondrial superoxide overprodn. activates poly(ADP-ribose) polymerase (PARP). PARP

activation, in turn, inhibits glyceraldehyde-3-phosphate dehydrogenase (GAPDH) activity, which activates at least three of the major pathways of hyperglycemic damage in endothelial cells. In this report, the authors show that hyperglycemia-induced overprodn. of superoxide by the mitochondrial electron transport chain activates the three major pathways of hyperglycemic damage found in aortic endothelial cells (activation of protein kinase C isoforms, hexosamine pathway flux, and advanced glycation endproduct [AGE] formation) by inhibiting GAPDH activity. Inhibition of GAPDH activity also activates the proinflammatory transcription factor NF-KB, which in aortic endothelial cells is PKC dependent. Hyperglycemia-induced GAPDH inhibition was found to be a consequence of poly(ADP-ribosyl)ation of GAPDH by poly(ADP-ribose) polymerase (PARP), which was activated by DNA strand breaks produced by mitochondrial superoxide overprodn. Both the hyperglycemia-induced decrease in activity of GAPDH and its poly(ADP-ribosyl)ation were prevented by overexpression of either uncoupling protein-1 (UCP-1) or manganese superoxide dismutase (MnSOD), which decrease hyperglycemia-induced superoxide. Overexpression of UCP-1 or MnSOD also prevented hyperglycemia-induced DNA strand breaks and activation of PARP. Hyperglycemia-induced activation of each of the pathways of vascular damage was abolished by blocking PARP activity with the competitive PARP inhibitors PJ34 or INO-1001. Thus, inhibition of PARP blocks hyperglycemia-induced activation of multiple pathways of vascular damage.

L2 ANSWER 17 OF 33 CA COPYRIGHT 2008 ACS on STN

TI Cloning and expression of ***adiponectin*** and its globular domain, and measurement of the biological activity in vivo

AB 3T3-L1 adipocytes produce the adipocyte complement related protein of 30 kD (ACRP30), which is exclusively expressed in differentiated adipocytes. Decreased expression of ACRP30 correlates with insulin resistance in mouse models of altered insulin sensitivity. ***Adiponectin*** , the human homolog of ACRP30, circulates in human plasma at high levels. Plasma ***adiponectin*** levels have been reported to be decreased in some insulin-resistant states, such as obesity and type II diabetes mellitus. The full-length ***adiponectin*** and its C-terminal globular head domain (gAdiponectin) were expressed in Escherichia coli and gAdiponectin was used to immunize a rabbit to obtain ***polyclonal*** antiserum with titer of 10,000. ***Adiponectin*** was detected in human plasma by Western blot anal. with gAdiponectin anti-serum or gACRP30 anti-serum. Injection in alloxan-treated rats with purified recombinant fusion ***adiponectin*** or gAdiponectin transiently abolished hyperglycemia, so ***adiponectin*** and gAdiponectin might have activity as a glucose lowering agent and potentially as a therapeutic for metabolic disease. All these results suggested that the recombinant protein had biol. activity, and provided a useful tool in further studies.

L2 ANSWER 18 OF 33 CA COPYRIGHT 2008 ACS on STN

TI Nucleic acid and polypeptide sequences for ***adiponectin*** and T-cadherin, receptor-ligand complexes, and therapeutic uses thereof for ***adiponectin*** -related diseases

AB The invention is directed to methods for identifying agents that mimic or modulate the interaction between ***adiponectin*** and its receptor, T-cadherin. The invention claims polynucleotide and polypeptide sequences for ***adiponectin*** and T-cadherin. In particular, the invention is directed to methods for mimicking or modulating the ***adiponectin*** -receptor interaction in order to treat diseases and disorders assocd. with a deficiency or overabundance of ***adiponectin*** . Such

diseases include obesity, anorexia nervosa, type I and type II diabetes, coronary artery disease and atherosclerosis. The invention also provides isolated ***adiponectin*** -T-cadherin complexes and methods for identifying polypeptides that interact with ***adiponectin*** . An in vivo dissocn. const. of 83 nM was detd. using recombinant mouse ***adiponectin*** lacking the signal sequence and T-cadherin. An anti-T-cadherin antibody reduced blood glucose levels in mice.

L2 ANSWER 19 OF 33 CA COPYRIGHT 2008 ACS on STN
TI ***Adiponectin*** and its receptors are expressed in bone-forming cells
AB ***Adiponectin*** has until now been considered to be synthesized and secreted exclusively by the adipose tissue, and is reported to influence energy homeostasis and insulin sensitivity. It is also known that body wt. is pos. correlated with increased bone mineral d. and decreased fracture risk. The mechanisms explaining this relation, however, are not completely understood. We report a link between ***adiponectin*** and bone homeostasis by demonstrating transcription, translation, and secretion of ***adiponectin*** , as well as expression of its receptors, AdipoR1 and AdipoR2, in bone-forming cells. We show that ***adiponectin*** and the receptors are expressed in primary human osteoblasts from femur and tibia. The phenotype of bone cells was confirmed by the high expression levels of alk. phosphatase, collagen type 1, osteocalcin, and CD44, and the formation of mineralization nodules. Immunostaining with ***monoclonal*** antibodies also demonstrated the presence of ***adiponectin*** in human osteosarcoma cells and normal osteoblasts. Both mRNA expression and secretion of ***adiponectin*** to the medium increased during differentiation of human osteoblasts in culture. The ***adiponectin*** mRNA level increases in osteoblasts cultured 3 and 7 days in the presence of dietary fatty acids and supplementation of culture medium with recombinant ***adiponectin*** enhances the proliferation of murine osteoblasts. The regulation and detailed function of ***adiponectin*** in bone still remains obscure, but our findings suggest a functional role in bone homeostasis. If so, ***adiponectin*** may provide an important signal linking fat and body wt. to bone d.

L2 ANSWER 20 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Latex reagent for ***adiponectin*** analysis, and ***adiponectin*** analysis method
AB A latex reagent for ***adiponectin*** anal. is provided, which comprises a suspension of latex particles carrying a substance (e.g., anti- ***adiponectin*** ***polyclonal*** antibody) capable of specifically binding with ***adiponectin*** . Also provided is a method for ***adiponectin*** anal., which comprises: (1) a step for obtaining a biol. liq. potentially contg. ***adiponectin*** ; and (2) a step for contacting the biol. liq. obtained in the step (1) as it is with a suspension of latex particles carrying a substance capable of specifically binding with ***adiponectin*** , and optically analyzing the resultant mixt. to det. the degree of agglutination of the latex particles. According to this latex reagent for ***adiponectin*** anal. and this ***adiponectin*** anal. method, it is not required to dil. or pretreat a biol. liq. as a test sample beforehand, and the anal. is rapidly and conveniently performed without limiting a measurement facility.

L2 ANSWER 21 OF 33 CA COPYRIGHT 2008 ACS on STN

TI ***Adiponectin*** specifically increased tissue inhibitor of metalloproteinase-1 through interleukin-10 expression in human macrophages
AB Vascular inflammation and subsequent matrix degrdn. play an important role in the development of atherosclerosis. The authors previously reported that ***adiponectin*** , an adipose-specific plasma protein, accumulated to the injured artery and attenuated vascular inflammatory response. Clin., high plasma ***adiponectin*** level was assocd. with low cardiovascular event rate in patients with chronic renal failure. The present study was designed to elucidate the effects of ***adiponectin*** on matrix metalloproteinases (MMPs) and tissue inhibitor of metalloproteinases (TIMPs) in human monocyte-derived macrophages. Human monocyte-derived macrophages were incubated with the physiol. concns. of human recombinant ***adiponectin*** for the time indicated.
Adiponectin treatment dose-dependently increased TIMP-1 mRNA levels without affecting MMP-9 mRNA levels. ***Adiponectin*** also augmented TIMP-1 secretion into the media, whereas MMP-9 secretion and activity were unchanged. Time course expts. indicated that TIMP-1 mRNA levels started to increase at 24 h of ***adiponectin*** treatment and were significantly elevated at 48 h. ***Adiponectin*** significantly increased interleukin-10 (IL-10) mRNA expression at the transcriptional level within 6 h and significantly increased IL-10 protein secretion within 24 h. Cotreatment of ***adiponectin*** with anti-IL-10 ***monoclonal*** antibody completely abolished ***adiponectin*** -induced TIMP-1 mRNA expression. Thus, ***adiponectin*** selectively increased TIMP-1 expression in human monocyte-derived macrophages through IL-10 induction. This study identified, for the first time, the ***adiponectin*** /IL-10 interaction against vascular inflammation.

L2 ANSWER 22 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Proteomic analysis of adipocyte differentiation: Evidence that .alpha.2 macroglobulin is involved in the adipose conversion of 3T3 L1 preadipocytes
AB Adipogenesis is an important aspect of energy homeostasis. Here we have used a differential proteome mapping strategy to identify intracellular proteins that are differentially expressed during adipose conversion of 3T3 L1 preadipocytes. Two-dimensional gel electrophoresis anal. identified 8 proteins that are induced following hormone-evoked differentiation. In addn., we found that a .alpha.2 macroglobulin fragment was abundantly present in 3T3 L1 preadipocytes, but was virtually undetectable in fully differentiated adipocytes. Metabolic radiolabeling with (35S)methionine and Northern blot anal. indicated that the intracellular .alpha.2 macroglobulin fragment in preadipocytes was derived from the extracellular culture medium, not de novo synthesis. Incubation of preadipocytes with an anti.alpha.2 macroglobulin ***polyclonal*** antibody caused depletion of the intracellular .alpha.2 macroglobulin fragments, and also enhanced spontaneous adipose conversion. These results suggest that intracellular .alpha.2 macroglobulin fragment inhibits adipocyte differentiation, and that hormone treatment induces differentiation at least in part by suppression of intracellular .alpha.2 macroglobulin activity in 3T3 L1 preadipocytes.

L2 ANSWER 23 OF 33 CA COPYRIGHT 2008 ACS on STN
TI ***Adiponectin*** suppresses proliferation and superoxide generation and enhances eNOS activity in endothelial cells treated with oxidized LDL
AB ***Adiponectin*** (also known as 30-kDa adipocyte complement-related protein or Acrp30) is an abundant adipocyte-derived plasma protein with anti-atherosclerotic and insulin-sensitizing properties. To investigate

the potential mechanism(s) of the vascular protective effect of ***adiponectin*** , the authors used cultured bovine endothelial cells (BAECs) to study the effect of recombinant globular ***adiponectin*** (gAd) on cellular proliferation and the generation of reactive oxygen species (ROS) induced by oxidized LDL (oxLDL). By RT-PCR, the authors found that BAECs preferentially express AdipoR1, the high-affinity receptor for gAd. Treatment of BAECs with oxLDL (10 .mu.g/mL) for 16 h stimulated cell proliferation by .apprx.60%, which was inhibited by co-incubation with gAd. Cell treatment with gAd also inhibited basal and oxLDL-induced superoxide release, and suppressed the activation of p42/p44 MAP kinase by oxLDL. The effects of gAd were blocked by a specific ***polyclonal*** anti- ***adiponectin*** antibody (TJ414). OxLDL-induced BAEC proliferation and superoxide release were inhibited by the NAD(P)H oxidase inhibitor diphenyleneiodonium (DPI), but not the eNOS inhibitor L-nitroarginine Me ester (l-NAME). Finally, gAd ameliorated the suppression of eNOS activity by oxLDL. These data indicate that gAd inhibits oxLDL-induced cell proliferation and suppresses cellular superoxide generation, possibly through an NAD(P)H oxidase-linked mechanism.

L2 ANSWER 24 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Hypoadiponectinemia is associated with impaired endothelium-dependent vasodilation
AB ***Adiponectin*** may have an antiatherogenic effect by reducing endothelial activation. We hypothesized that plasma ***adiponectin*** levels were correlated with endothelial function. Plasma ***adiponectin*** level was detd. by an inhouse RIA assay using a rabbit ***polyclonal*** antibody in 73 type 2 diabetic patients and 73 controls. Endothelium-dependent and independent vasodilation of the brachial artery was measured by high-resoln. vascular ultrasound. Plasma ***adiponectin*** level was lower in diabetic patients than in controls (4.73.+-.1.96 vs. 7.69.+-.2.80 .mu.g/mL, resp.; P < 0.001), and they also had impaired endothelium-dependent (5.6.+-.3.6 vs. 8.6.+-.4.5%, resp.; P < 0.001) and -independent vasodilation (13.3.+-.4.9 vs. 16.5.+-.5.6%, resp.; P < 0.001). Plasma ***adiponectin*** correlated with endothelium-dependent vasodilation in controls (P = 0.02) and diabetic patients (P = 0.04). On general linear-model univariate anal., brachial artery diam., the presence of diabetes, plasma ***adiponectin*** , and high-d. lipoprotein were significant independent determinants of endothelium-dependent vasodilation. In vitro expts. showed that endothelial cells expressed ***adiponectin*** receptors, and ***adiponectin*** increased nitric oxide prodn. in human aortic endothelial cells. In conclusion, low plasma ***adiponectin*** level is assocd. with impaired endothelium-dependent vasodilation, and the assocn. is independent of diabetes mellitus. ***Adiponectin*** may act as a link between adipose tissue and the vasculature.

L2 ANSWER 25 OF 33 CA COPYRIGHT 2008 ACS on STN
TI ***Monoclonal*** antibody against ***adiponectin***
AB The authors disclose prepn. of a ***monoclonal*** antibody that recognizes ***adiponectin*** . The present invention provides a measuring method for concn. of the ***adiponectin*** in a sample, esp. blood with the ***monoclonal*** antibody, thereby rendering diagnosis of ***adiponectin*** -assocd. diseases such as Type II diabetes and obesity.

L2 ANSWER 26 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Plasma resistin concentrations measured by enzyme-linked immunosorbent assay using a newly developed ***monoclonal*** antibody are elevated in individuals with type 2 diabetes mellitus
AB Resistin is an adipocyte-derived peptide that might play a role in obesity and insulin resistance. However, its role in humans is largely unclear. Although many studies have measured the expression of human resistin in tissues, the circulating concns. of resistin and its relation to metabolic parameters in humans are unknown. We developed an ELISA for human resistin and measured plasma concns. in aged individuals with or without type 2 diabetes mellitus. To validate the results of plasma resistin concns. in our subjects, plasma ***adiponectin*** concns. were also detd., which were higher in nondiabetic subjects than in type 2 diabetic patients and correlated with the homeostasis model assessment for insulin resistance (HOMA-IR). Log-transformed plasma resistin concns. (log-resistin) were higher in diabetic patients compared with normal individuals (0.50 .+-. 0.39 vs. 0.28 .+-. 0.51 ng/mL), and this difference was significant after controlling for gender and body mass index. Log-resistin did not show a significant correlation with HOMA-IR, waist circumference, body mass index, blood pressure, or total cholesterol. The plasma glucose concn. was an independent factor assocd. with log-resistin. In conclusion, plasma resistin concns. are elevated in patients with type 2 diabetes, but are not assocd. with insulin resistance or obesity.

L2 ANSWER 27 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Method for diagnosing or monitoring carbohydrate metabolism abnormality
AB A method is provided for diagnosing an insulin resistance-related adult disease such as type 2 diabetes at its early stage by quant. measuring ***adiponectin*** (GBP28) in a sample using a ***monoclonal*** antibody capable of measuring GBP28. A method is also provided for monitoring the therapeutic effect of a therapeutic agent for type 2 diabetes.

L2 ANSWER 28 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Human cDNA sequences and their encoded proteins and diagnostic and therapeutic uses
AB Disclosed herein are 62 cDNA sequences that encode novel human polypeptides that are members of the following protein families: trypsin, germline oligomeric matrix protein, neuromedin U25, caldecrin, neural cell adhesion protein, ADAMTS 12, CASPR4, ADAMS-TS3, gliacolin, aminopeptidase N, ***adiponectin*** , trypsin III, tissue kallikrein, .beta.-transforming growth factor, diphthamide synthesis protein, WECHE lungkine, ADAM-TS7, palmitoyl-protein thioesterase-2I, betacellulin, small inducible cytokine A23, granulocyte colony-stimulating factor, platelet basic protein 2, brain natriuretic peptide, serine protease, acyl-CoA-binding protein, elastase IV, collagen, viral receptor, and cathepsin L2. Also disclosed are polypeptides encoded by these nucleic acid sequences, and antibodies, which immunospecifically-bind to the polypeptide, as well as derivs., variants, mutants, or fragments of the aforementioned polypeptide, polynucleotide, or antibody. The invention further discloses therapeutic, diagnostic and research methods for diagnosis, treatment, and prevention of disorders involving any one of these novel human nucleic acids and proteins.

L2 ANSWER 29 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Influences of ionomycin, dibutyryl-cycloAMP and tumour necrosis factor-alpha on intracellular amount and secretion of ***apM1*** in

differentiating primary human preadipocytes

AB 3T3-L1-adipocytes produce the adipocyte complement related protein of 30 kD (Acrp30), which is also designated as AdipoQ. In order to study the expression and secretion of the human homolog of this protein, ***apM1*** (adipose Most abundant gene transcript 1, also named gelatin-binding protein of 28 kD [GBP28] or ***adiponectin***), a ***polyclonal*** antibody was produced. Both expression and secretion can be detected beginning with day 4 after induction of differentiation. The amt. of expressed ***apM1*** correlates with the specific activity of the differentiation marker glycerol-3-phosphate dehydrogenase. Secretion of ***apM1*** is increased by the addn. of ionomycin. Both the nonhydrolysable dibutyryl-cycloAMP and tumor necrosis factor alpha reduce the expression and secretion of ***apM1*** .

L2 ANSWER 30 OF 33 CA COPYRIGHT 2008 ACS on STN

TI ***Adiponectin*** , a new member of the family of soluble defense collagens, negatively regulates the growth of myelomonocytic progenitors and the functions of macrophages

AB The authors investigated the functions of ***adiponectin*** , an adipocyte-specific secretory protein and a new member of the family of sol. defense collagens, in hematopoiesis and immune responses. ***Adiponectin*** suppressed colony formation from colony-forming units (CFU)-granulocyte-macrophage, CFU-macrophage, and CFU-granulocyte, whereas it had no effect on that of burst-forming units-erythroid or mixed erythroid-myeloid CFU. In addn., ***adiponectin*** inhibited proliferation of 4 of 9 myeloid cell lines but did not suppress proliferation of erythroid or lymphoid cell lines except for one cell line. Thus, ***adiponectin*** predominantly inhibits proliferation of myelomonocytic lineage cells. At least one mechanism of the growth inhibition is induction of apoptosis because treatment of acute myelomonocytic leukemia lines with ***adiponectin*** induced the appearance of subdiploid peaks and oligonucleosomal DNA fragmentation. Aside from inhibiting growth of myelomonocytic progenitors, ***adiponectin*** suppressed mature macrophage functions. Treatment of cultured macrophages with ***adiponectin*** inhibited their phagocytic activity and their lipopolysaccharide-induced prodn. of tumor necrosis factor .alpha.. Suppression of phagocytosis by ***adiponectin*** is mediated by one of the complement C1q receptors, C1qRp, because this function was completely abrogated by the addn. of an anti-C1qRp ***monoclonal*** antibody. Thus, ***adiponectin*** is an important neg. regulator in hematopoiesis and immune systems and it may be involved in ending inflammatory responses via its inhibitory functions.

L2 ANSWER 31 OF 33 CA COPYRIGHT 2008 ACS on STN

TI The human apM-1, an adipocyte-specific gene linked to the family of TNF's and to genes expressed in activated T cells, is mapped to chromosome 1q21.3-q23, a susceptibility locus identified for familial combined hyperlipidaemia (FCH)

AB The human adipocyte-specific apM-1 gene encodes a secretory protein of the adipose tissue that has been suggested to play a role in the pathogenesis of obesity. The regulation of apM-1 was studied along adipocyte differentiation. While apM-1-mRNA and apM-1 protein were absent in preadipocytes and in 48 h differentiated adipocytes, they were found upregulated from day 4 to day 9 of adipocyte differentiation as shown by RNase protection assay and Western blot anal. These data indicate that apM-1 may be a late marker of adipocyte differentiation. In human sera apM-1 protein is also detectable by Western blots using a

polyclonal antibody raised against a synthetic peptide sequence of
the human apM-1. The genomic structure of the human apM-1 gene together with a total of 2.7 kb of the 5'-flanking region with putative transcription factor binding sites is presented. Interestingly, sequence comparisons link the apM-1 gene to the family of TNF's and to genes expressed in activated T-cells. The chromosomal localization of apM-1 was investigated by FISH and mapped to human chromosome 1q21.3-1q23, a region that was identified as a susceptibility locus for Familial Combined Hyperlipidemia (FCH) and polygenic NIDDM. These data and the chromosomal localization on chromosome 1q21.3-q23 raises the possibility that apM-1 as an adipocyte-specific secretory protein may play a role in the pathogenesis of FCH and assocd. insulin resistance. Exon- and intron-specific primer sequences are presented as a basis for mutation screening of patients affected with FCH. (c) 1999 Academic Press.

L2 ANSWER 32 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Paradoxical decrease of an adipose-specific protein, ***adiponectin***, in obesity
AB We isolated the human adipose-specific and most abundant gene transcript, ***apM1*** (Maeda, K.; et al., 1996). The ***apM1*** gene product was a kind of sol. matrix protein, which we named ***adiponectin*** . To quantitate the plasma ***adiponectin*** concn., we have produced ***monoclonal*** and ***polyclonal*** antibodies for human ***adiponectin*** and developed an ELISA system. ***Adiponectin*** was abundantly present in the plasma of healthy volunteers in the range from 1.9 to 17.0 mg/mL. Plasma concns. of ***adiponectin*** in obese subjects were significantly lower than those in non-obese subjects, although ***adiponectin*** is secreted only from adipose tissue. The ELISA system developed in this study will be useful for elucidating the physiol. and pathophysiol. role of ***adiponectin*** in humans. (c) 1999 Academic Press.

L2 ANSWER 33 OF 33 CA COPYRIGHT 2008 ACS on STN
TI Fat tissue-specific factor ***apM1*** for inhibiting smooth muscle proliferation and its use for the diagnosis and treatment of arteriosclerosis
AB Described is a pharmaceutical compn. contg. fat tissue-specific factor ***apM1*** (adipose most abundant gene transcript 1) that is able to inhibit the smooth muscle proliferation and the expression of adhesion mols. in vascular endothelial cells, and its use for prevention and treatment of arteriosclerosis and of re-narrowing after vascular surgery. Also described is a (***monoclonal***) antibody to ***apM1*** , its use for diagnosis of arteriosclerosis, and a diagnostic kit contg. the antibody. Prepn. of ***apM1*** by expression of the encoding gene in Escherichia coli; prepn. of rabbits ***polyclonal*** antibodies and mouse ***monoclonal*** antibody to ***apM1*** ; immunoassay of ***apM1*** in human blood samples with the antibodies; and its inhibitory effects on human smooth muscle cells and adhesion mols. such as VCAM-1 (vascular cell adhesion mol.-1), ELAM (endothelial leukocyte adhesion mol.), and ICAM-1 (intercellular adhesion mol.-1) were also shown.

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L2 ANSWER 25 OF 33 CA COPYRIGHT 2008 ACS on STN

AN 140:252318 CA <<LOGINID::20080330>>
ED Entered STN: 08 Apr 2004
TI ***Monoclonal*** antibody against ***adiponectin***
IN Youn, Byung-soo; Yang, Young-soo; Lee, Nam-seok; Yu, Kang-yeol; Youn, Moon-yeon; Park, Hong-je; Min, Sung-shik; Jeoung, Jae-jun
PA Komed Co., Ltd., S. Korea
SO PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C07K016-18
CC 15-3 (Immunochemistry)
Section cross-reference(s): 2, 14
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004022596	A1	20040318	WO 2003-KR1213	20030619
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	KR 2004022384	A	20040312	KR 2003-38880	20030616
	AU 2003244237	A1	20040329	AU 2003-244237	20030619
PRAI	KR 2002-53427	A	20020905		
	KR 2003-38880	A	20030616		
	WO 2003-KR1213	W	20030619		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 2004022596	ICM	C07K016-18
	IPCI	C07K0016-18 [ICM,7]
	IPCR	A61K0039-00 [N,C*]; A61K0039-00 [N,A]; C07K0016-18 [I,C*]; C07K0016-18 [I,A]
	ECLA	C07K016/18
KR 2004022384	IPCI	C07K0016-18 [ICM,7]
AU 2003244237	IPCI	C07K0016-18 [ICM,7]
	IPCR	A61K0039-00 [N,C*]; A61K0039-00 [N,A]; C07K0016-18 [I,C*]; C07K0016-18 [I,A]

AB The authors disclose prepn. of a ***monoclonal*** antibody that recognizes ***adiponectin*** . The present invention provides a measuring method for concn. of the ***adiponectin*** in a sample, esp. blood with the ***monoclonal*** antibody, thereby rendering diagnosis of ***adiponectin*** -assocd. diseases such as Type II diabetes and obesity.
ST ***monoclonal*** antibody ***adiponectin***
IT Hybridoma
(B-cell, KCTC 10289BP; for antibodies to ***adiponectin***)
IT Antibodies and Immunoglobulins
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)

(IgG1, ***monoclonal*** ; to human ***adiponectin***)
IT Cytokines
RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(***adiponectin*** ; prepn. of ***monoclonal*** antibodies to)
IT Diagnosis
(diabetes mellitus; ***monoclonal*** antibodies to human ***adiponectin*** for)
IT Immunoassay
(enzyme-linked immunosorbent assay; ***monoclonal*** antibodies for detection of human ***adiponectin***)
IT Test kits
(for immunoassay of ***adiponectin***)
IT Antibodies and Immunoglobulins
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(immunoadhesins; of human ***adiponectin*** for prepn. of ***monoclonal*** antibodies)
IT Blood analysis
(***monoclonal*** antibodies for detection of human ***adiponectin***)
IT Obesity
(***monoclonal*** antibodies for detection of human ***adiponectin*** in)
IT Bos taurus
Capra
Equus caballus
Human
Mammalia
Mus
Oryctolagus cuniculus
Rattus
(***monoclonal*** antibodies to ***adiponectin*** of)
IT Diagnostic agents
(***monoclonal*** antibodies to human ***adiponectin*** for)
IT Diabetes mellitus
(non-insulin-dependent; ***monoclonal*** antibodies for detection of human ***adiponectin*** in)
IT Fusion proteins (chimeric proteins)
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(of human ***adiponectin*** for prepn. of ***monoclonal*** antibodies)
IT Diagnosis
(serodiagnosis; ***monoclonal*** antibodies to human ***adiponectin*** for)
IT 670342-21-9 670342-22-0
RL: PRP (Properties)
(unclaimed nucleotide sequence; ***monoclonal*** antibody against ***adiponectin***)
IT 670222-47-6 670222-48-7 670222-49-8 670222-50-1 670342-23-1 670342-25-3
RL: PRP (Properties)
(unclaimed sequence; ***monoclonal*** antibody against ***adiponectin***)
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE

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(5) Sanofi-Synthelabo; EP 1002865 A1 2000 CA

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(FILE 'HOME' ENTERED AT 19:39:11 ON 30 MAR 2008)

FILE 'CA' ENTERED AT 19:39:23 ON 30 MAR 2008
L1 3952 S ADIPONECTIN OR APM1
L2 33 S L1 AND (MONOCLONAL OR POLYCLONAL)
L3 1 S L2 AND LATEX PARTICLES

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
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